

Hypocalcemic Seizures in an African Grey Parrot

Lawrence J. McDonald

A six-year-old African grey parrot (*Psittacus erithacus erithacus*), presumed to be a male although surgical sexing was not done, was presented to the hospital on an emergency basis. The presenting complaint was three seizures in the past three days. Prior to this presentation the bird had had two other seizure attacks in the preceding month. The client had owned this bird for four years, and had not detected any previous problems. Nine other birds lived in the home: a female African grey, six Amazon parrots and two conures. All of these other birds were fine according to the owner, and none of them had ever had a detectable seizure.

After the seizures at home, the bird returned to normal within a few minutes. He ate well, had no regurgitation, diarrhea, cough, or sneeze. The diet consisted of peanuts and sunflower seeds, an assorted seed mix, fresh vegetables (carrots, lettuce, spinach, and broccoli), and fresh fruits (apples, oranges, and grapes), all *ad lib*. No vitamin or mineral supplementation was given, and the client remarked that the bird's favorite food seemed to be peanuts.

The bird was well fleshed (448 grams) and had good feather, beak and nail quality. No clinical abnormalities could be found. The neurological exam appeared normal; however, handling the bird was not as difficult as is usual with African grey parrots. The bird was then hospitalized, and blood for a routine hematological profile was obtained from both the hospitalized male bird and the female African grey parrot that currently lived in the home (Table 1). Bacterial culture and sensitivity was conducted on a cloacal sample taken from the subject bird.

Until the clinical pathology results became available, emergency treatment consisted of 25 mg calcium gluconate (calcium gluconate injection B.P., Glaxo Laboratories, Toronto, Ontario) given subcutaneously in 5 mL physiological saline (sodium chloride injection U.S.P., Abbott Laboratories, Montreal, Quebec), distributed in three sites over the pectoral musculature, and Injacom 100 + B complex (Hoffman LaRoche, Nutley, New Jersey) 0.06 mL diluted in 1 mL physiological saline subcutaneously (equals 6000 IU vitamin A and 600 IU vitamin D3). The bird was placed in a hospital cage for observation and was fed a seed mix. Spectinomycin oral liquid (Syntex Agribusiness, Mississauga, Ontario) (15 drops in 120 mL water), calcium (Calcium Sandoz syrup 5 mL = 110 mg, Sandoz Canada, Dorval, Quebec) (1 mL in 30 mL water), and WinStress powder (WinStress, Winthrop Animal Health Products, Aurora, Ontario) (1/8 tsp in 600 mL water) were added to the bird's drinking water.

By the following day the bird was eating well, passing normal stools, and was much stronger and more aggressive. On reviewing the clinical pathology results (Table 1), the most probable cause of the seizures appeared to be hypocalcemia. Subsequently the treatment consisted of the previously discussed oral medications, with the exception of the spectinomycin oral liquid because of the resistance of *Enterobacter cloacae* to spectinomycin. Spectinomycin was replaced with carbenicillin (Geopen 500 mg, Pfizer, Kirkland, Quebec) 95.5 grams added to 120 mL water with 2 to 3 drops of Sucaryl (cyclamate, Abbott Laboratories, Montreal, Quebec) added to improve palatability. Oral medication was chosen rather than parenteral due to the increasingly fractious nature of the bird. The patient was discharged from the hospital on the same dose of Calcium Sandoz, WinStress and carbenicillin with Sucaryl. Seven days later the bird appeared to be doing well; however, due to costs the owner declined to have the blood profile repeated. Carbenicillin was discontinued at that time, but the client was instructed to continue with Calcium Sandoz (1 mL in 30 mL water) and WinStress (1/8 tsp in 600 mL water), both made up fresh daily and administered for the life of the bird. The client was also instructed to try to correct the bird's diet; this involved limiting the consumption of peanuts and sunflower seeds each day and feeding a variety of seeds, feeding foods high in calcium, such as cheese and yogurt, and offering more fresh fruits and vegetables.

Calcium and phosphorus homeostasis is a complex interaction of hormones (parathyroid hormone (PTH), calcitonin, and vitamin D3 and its metabolites) and body organs (bones, gastrointestinal tract, kidney, and liver).

Production of PTH is influenced by two major regulators: ionized calcium (Ca^{++}) (1,2) and cyclic 3',5'-adenosine monophosphate (cAMP) (2). As Ca^{++} decreases or cAMP increases, the parathyroid gland produces more PTH. Petrak (1) and Stunkard (3) also suggest that in poultry PTH and estrogen have a synergistic effect on ionized calcium levels.

Target organs of PTH are bones, kidney and gastrointestinal tract. Under the influence of PTH there is increased mobilization of calcium from bone into blood. This occurs due to increased osteolytic osteolysis and osteoclastic bone resorption (2). Parathyroid hormone causes increased renal absorption of calcium and increased excretion of phosphorus by the renal tubular epithelium.

Vitamin D3 can be absorbed from food, and perhaps made by the action of sunlight on the uropygeal gland secretions which the bird consumes when preening (4). Once vitamin D3 (cholecalciferol) is absorbed from the intestinal tract it is metabolized in the liver and kidney to two compounds: 1,25 dihydroxycholecalciferol and 24,25 dihydroxycholecalciferol (4,5). If hypocalcemia

TABLE 1
Hematological and Biochemical Findings in a Male
African Grey Parrot with Seizures, and a Normal
Female African Grey Parrot

	Male	Female	Normal Values for African Grey Parrots (8,9)
WBC ($\times 10^9/L$)	6-8	3-4	5-11
PCV (L/L)	0.50	0.45	0.43-0.55
Plasma protein (g/L)	37	35	30-50
Heterophils ($\times 10^9/L$)	4.7	1.0	2.2-8.2
Lymphocytes ($\times 10^9/L$)	2.1	2.4	1.0-5.5
Monocytes ($\times 10^9/L$)	0.1	0.1	0-0.3
Eosinophils ($\times 10^9/L$)	0.1	0	0-0.2
Basophils	0	0	0-0.5
Thrombocytes	normal	normal	
Polychromasia	slight	slight	
Anisocytosis	slight	slight	
AST (U/L)	724	198	100-350
Uric acid ($\mu\text{mol/L}$)	390	450	240-590
LDH (U/L)	3408	313	150-450
Glucose (mmol/L)	21	16	10-19
Calcium (mmol/L)	1.4	2.2	2.0-3.2
CK (U/L)	10587	350	100-350

occurs, PTH is secreted which increases the synthesis of 1,25 dihydroxycholecalciferol, which in turn promotes absorption of calcium from the intestinal tract and mobilization of calcium and phosphorus from the bones.

Calcitonin is produced in the ultimobranchial gland in birds (4). Its primary action is to oppose the action of PTH on kidney and bone. If there is a pathological process in a bird's gastrointestinal tract, parathyroid gland or bones, a hypocalcemic state can result.

The other obvious cause of hypocalcemia is deficient intake of calcium and vitamin D3. A deficiency of vitamin D3 results in hypocalcemia after 14 days, even if the diet contains 1% calcium (4). Continued deficiency ultimately results in osteomalacia. The normal dietary Ca:P ratio should approach 1.5:1 (4,6), which approximates a diet containing 1% calcium and 0.7% phosphorus. Most seeds that are eaten by caged birds are low in calcium (4,6). Peanuts and sunflower seeds are particularly deleterious to calcium homeostasis, for not only do they only contain about 20% of the calcium requirement of a nonlaying bird (3), but they also have a high fat content. This fat may combine in the gastrointestinal tract (3,4) with calcium to form insoluble soaps (3,4). Both of these situations can lead to nutritional "metabolic bone disease". This term applies to the compensatory hypersecretion and hyperplasia of the parathyroid glands in an attempt to maintain normocalcemia.

Finally, in the African grey parrot (*Psittacus erithacus erithacus*) and the Timneh grey parrot (*Psittacus erithacus timneh*) (5,7,8), a hypocalcemic syndrome has been documented. It is usually seen in young birds (two- to five-years-old) but has been reported in a ten-year-old bird. The birds have a history of fainting or seizures, often initiated by excitement. Blood calcium levels are consistently below normal (i.e. 6 mg/dL with the normal being 8 to 13 mg/dL) (9), and have been recorded as low as 2 to 4 mg/dL. On histopathology parathyroid glands are

enlarged and there are severe degenerative changes. Vacuolation of adrenocortical cells is also present. Cortical bone sections, however, demonstrate no calcium loss or thinning as would be expected in a hypocalcemic state.

The authors (5,7) then speculate on possible etiologies. One theory is that perhaps African grey parrots cannot mobilize skeletal calcium in the face of hypocalcemia. Perhaps viral damage to the parathyroid glands is necessary to trigger or enhance this inability, for apparently not all African grey parrots manifest this problem. A second theory involves renal function. This hypothesis assumes a loss of calcium through the kidney; however, a "trigger" for this phenomenon is unknown. The third and final hypothesis is that perhaps the syndrome is entirely dietary, with the concurrent inability to mobilize skeletal calcium. Obviously a definitive pathogenesis is lacking, but the syndrome is frequently seen (7) in these two species of birds.

The clinical pathology results raise several major doubts. First, that the most probable cause of the seizures was hypocalcemia. Other potential causes of seizures (6) in birds include hypoglycemia, toxicosis (lead, mercury), infections, and epilepsy. This bird was not hypoglycemic. The prompt response to calcium therapy, together with no recurrence of seizures over a sixteen month period would tend to rule-out epilepsy. The possibility of a concurrent bacteremia must be considered due to the Enterobacteriaceae cloacal culture. Enterobacteriaceae frequently cause disease in pet birds (8), and birds seem prone to develop bacteremia. Thus, the initiation of the seizures by infection plus stress, as manifested by the increase in heterophils and glucose (Table 1), cannot be ruled out. The female bird, living in the same home and fed the same diet as the subject bird for four years, did not manifest hypocalcemia. Perhaps this would introduce some skepticism as to the hypothesis (5,7) of a virus damaging the parathyroid gland of the male bird.

Circumstantial evidence would suggest that the male bird was affected by the African grey parrot hypocalcemia syndrome because the female, which was on the same relatively calcium-deficient diet, did not manifest hypocalcemia.

Prophylactic measures to prevent the hypocalcemic seizure syndrome in African grey and Timneh grey parrots should include vitamin D3 supplementation and periodic determinations of blood calcium levels. A calcium replete diet should obviously be provided; client counselling in this area is very important. I have seen three cases in the past year, but this is the only patient to survive the initial hypocalcemic emergency situation. Consequently, with all African grey patients, I now stress client education and provide in-depth diet sheets in an attempt to avoid a crisis situation.

References

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